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appearance, he completely lost the power of locomotion, and possessed no voluntary power but that of turning his head from side to side, and of moving his toes. The skin remained quite sensible to the touch, the functions of the primae viae and urinary organs continued natural, and he had neither headache nor giddiness".

Nearly four months from the beginning of his illness his clinical state was more or less unchanged. "... he could not without a great effort move even in a slight degree either the upper or the lower extremities, but he could readily move his head and toes. The functions of the organs of sense were entire. He had no difficulty in voiding water: his appetite was good, and he had a daily alvine discharge. His pulse was natural, he had no febrile symptoms, and he was not much reduced in flesh. He stated, however, that for some time previous to this illness, the abdomen had gradually become full and tense, and several days previous to the attack he complained of diarrhoea, at which period he had travelled on the outside of a coach and caught cold".

Wardrop treated his patient by purging and in about three weeks improvement began, with movement being restored to the fingers and then the arm and legs. After four months he could walk without assistance and he could write. "From this period he continued perfectly well," Wardrop concluded, "and I saw him several years afterwards in the enjoyment of good health, having never had any return of the symptoms."

It is difficult to avoid the diagnosis of acute polyradiculoneuropathy in this patient. He probably displayed at least seven of the diagnostic criteria formulated by the National Institute of Neurological and Communicative Disorders and Stroke,² and there was the history of gastrointestinal illness. Wardrop could not have described loss of tendon jerks because their significance was not to be realised until about 40 years later.³ Lumbar puncture for CSF examination was not introduced for another 57 years.⁴ Landry's original description of "ascending paralysis" was in 1859. The first report of the CSF findings by Guillain, Barré, and Strohl was published in 1916, and this was amplified later.⁴

It would seem that Wardrop should have credit for the earliest clinical description of this disease.

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TETRAHYDROAMINOACRIDINE AND THE CENTRAL ANTICHOLINERGIC SYNDROME

SIR,—I was intrigued by your editorial (Jan 17, p 139) on the cholinergic treatment of Alzheimer's disease via oral tetrahydroaminoacridine (THA). Before the availability of short-duration, non-depolarising muscle relaxants THA was often used during general anaesthesia to prolong the very brief action of suxamethonium to 15-20 min. Nowadays parenteral THA is only available on a named patient basis, and oral THA has never been available in the UK. When I regularly used THA to prolong the action of suxamethonium I formed the clinical impression that confused and elderly patients could sometimes be strikingly brighter mentally in the postoperative period for a day or so. It is interesting to speculate, in the light of your editorial, whether this increased mental alertness might have been a consequence of restored levels of cholinergic activity in the brain, though there are other possible explanations.

The antimuscarinic properties of hyoscine and atropine make these drugs valuable premedicants before the induction of general anaesthesia. Hyoscine calms the central nervous system except in elderly patients, in whom unacceptable agitation and confusion can result. For this reason anaesthetists traditionally eschew the use of hyoscine in patients over the age of 60 or 65. Older patients are usually given atropine instead but perhaps a better choice is glycopyrrolate, which has only peripheral anticholinergic effects in

premedication dosage. Apart from these considerations there is generally little awareness amongst anaesthetists that they might be harming patients by the unrestricted use of anticholinergic drugs.

The need to recognise that the central anticholinergic syndrome (CAS)¹ can be precipitated by anaesthetists has been emphasised by Rupreht and Dworacek.² The mental impairment associated with CAS ranges from simple memory disturbance to delirium and coma. The prescribed treatment is the careful intravenous administration of physostigmine salicylate. I have found this treatment effective in patients with arteriopathy who have remained agitated and confused 60-120 min postoperatively after apparently adequate reversal of muscle relaxation and uneventful general anaesthesia. The neostigmine used to reverse muscle relaxant drugs does not cross the blood-brain barrier, unlike physostigmine, and would not be expected to remedy the CAS. There is clearly an important differential diagnosis to be made by the anaesthetist between the confused agitation of incomplete reversal of the muscle relaxant, the continued effects of other anaesthetic agents, and the onset of CAS. Recognisable mental changes associated with CAS can last a day or much longer in susceptible patients so that the careful administration of physostigmine in these patients is justified. It would be interesting to know whether THA, which can also cross the blood-brain barrier, might be a suitable alternative parenteral therapy. Peripheral muscarinic side-effects caused by both drugs can be blocked with glycopyrrolate. The discomforts and possible hazards of administering anticholinesterases parenterally make it essential to consider very carefully whether a patient has CAS before beginning treatment.

Further investigation of CAS is urgently required before it becomes deeply engrained in the literature of anaesthesia. One objection to the concept of CAS as a complication of anaesthesia is that there is a multitude of neurotransmitters involved in the ascending pathways that alert the cerebral cortex, all of which may be altered by the complex effects of general anaesthesia.

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ALZHEIMER'S DISEASE AND TETRAHYDROAMINOACRIDINE

SIR,—Your Jan 17 editorial, when referring to the trial of Summers et al,¹ states that, apart from tetrahydroaminoacridine, the patients took 1200 mg of lecithin daily. The paper states that all patients were asked to take nine or more capsules of commercial lecithin (1200 mg per capsule) or its equivalent per day—ie, a daily intake of at least 10.80 g lecithin.

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EICOSAPENTAENOIC ACID IN FAT

SIR,—The health effects of the ω 3-polyunsaturated fatty acid, eicosapentaenoic acid (EPA; 20:5[n-3]), are of great interest. Epidemiological investigations, however, are hampered by problems in determining long-term dietary intake of EPA. Dietary survey methods are imprecise and data on EPA in foods are scarce. For the assessment of long-term individual intake of linoleic acid (C18:2[n-6]), fat tissue microbiopsy has proved a reliable alternative to survey methods,¹ and a similar method for EPA would be useful. Dr Wood and colleagues, in their paper on tissue fatty acids and coronary heart disease (Jan 24, p 177), state that EPA is not present in adipose tissue. We have now found that EPA can be reliably detected in fat tissue microbiopsy specimens by capillary gas/liquid chromatography (figure). In three healthy volunteers (a

Detection of EPA in adipose tissue.

Peaks identified by comparison with pure standards. EPA (20:5[n-3]) content is 0.08% fatty acid methyl esters; detection limit about 0.01%. Inset compares putative EPA peak (lower) with peak when standard EPA (donated by Dr G. Hornstra) was added to the biopsy fatty acids.

Fatty acid methyl esters prepared from an 11 mg microbiopsy specimen² were injected into a Packard gas chromatograph, split ratio 30:1, carrier gas hydrogen 130 kPa, column WCOT fused silica 50 m × 0.22 mm, coated with 0.2 µm 'CP-Sil 88' (Chrompack, Middelburg, Netherlands), with oven temperature 210°C, FID detector, and injector 250°C.

female student, a female technician, and a male staff member) we found EPA levels of 0.07, 0.04, and 0.08 g per 100 g fatty acid methyl esters, respectively. None of them ate a lot of fish.

Whether fat tissue EPA is a valid measure of EPA consumption remains to be established.

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BODYBUILDER'S PSYCHOSIS

SIR,—Anabolic steroids are sometimes used by athletes seeking gains in muscle size and strength.^{1,2} Although the medical effects and risks of these drugs are well documented, their psychiatric effects are largely unexplored.³⁻⁵

We have treated two men who required hospital admission for psychotic episodes apparently associated with use of anabolic steroids. A 40-year-old man had been prescribed methyl-testosterone 10 mg twice daily for idiopathic impotence. Within 2 weeks he had symptoms meeting DSM-III criteria⁶ for major depression, together with delusions of reference and visual and auditory hallucinations. A 22-year-old construction worker and bodybuilder took two 8-week courses of methandrostenolone 15 mg daily. After the second course he had severe depressive symptoms. Although the depression gradually lifted after several months prominent paranoid and religious delusions ensued.

Extensive medical and neuroendocrine workup of both patients was unremarkable. Both responded well to neuroleptics and were able to discontinue these medications permanently after several weeks. Neither patient reported any serious psychopathology before the index episode. Both, with no further exposure to steroids, have remained psychiatrically normal during more than two years' follow-up. Thus, anabolic steroids emerge as a likely causative factor in both cases.

Intrigued by these observations, we interviewed thirty-one other anabolic steroid users (twenty-nine men), recruited by

advertisements at gymnasia in the Boston and Los Angeles areas. Psychiatric diagnoses were made using a structured clinical interview.⁷ Three men had had psychotic symptoms with steroids: one reported auditory hallucinations of voices and noises lasting 5 weeks; another described delusions of reference and possible thought broadcasting; a third reported paranoid delusions. At least four others had "subthreshold" psychotic symptoms,⁷ including referential thinking, paranoid jealousy, and grandiose beliefs. For example, one man, while on steroids, believed that he could not be injured if he fell from a third-floor window. All seven experienced such symptoms only when taking orally active 17-alkylated steroids (methandrostenolone, oxandrolone, and/or oxymetholone), alone or in combination with parenteral preparations. None described any psychotic symptoms, even at the subthreshold level, at any time before steroid use.

Equally impressive was the observation that four interviewees met criteria⁶ for manic episodes while taking anabolic steroids, and five had major depression, either during or just after a course of the drugs. Manic-like behaviours were prominent; one 23-year-old twice bought expensive cars during courses of steroids and then was forced to sell them afterwards. Another subject, taking methandrostenolone, deliberately drove an old car into a tree at 60 km/h while a friend videotaped it. A third man, when "cut up" by another driver in Boston, pursued and cornered the offender and smashed his windshield with a crowbar. None of these subjects described comparable behaviours when not using steroids.

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BACK PAIN AND TESTICULAR GERM CELL TUMOURS

SIR,—Dr Taylor (Feb 28, p 515) is correct in stating that germ cell tumours with no evidence of a testicular primary are rare, but patients are notoriously shy about reporting testicular abnormalities¹ and even locally advanced tumours could escape notice during a routine orthopaedic examination. Oncologists have learned that back pain in cancer patients can indicate relapse in the retroperitoneal lymph nodes.² Relief of pain is then a useful clinical marker of response to treatment.

We have found that severe, persistent back pain is the presenting feature of many malignancies in young people, including lymphoma, testicular tumours, and carcinoma of the cervix (to be published). Retroperitoneal lymphadenopathy can usually be demonstrated in these patients by simple ultrasound examination. It is important that the association between back pain and malignant lymphadenopathy is more widely recognised so that these potentially curable tumours are detected early.

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